In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: December 20, 2023

LARRY J. PARKER,	*	PUBLISHED
Petitioner,	*	No. 20-411V
v.	*	Special Master Nora Beth Dorsey
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	Ruling on Entitlement; Pneumococcal Conjugate ("Prevnar 13") Vaccine; Guillain-Barré Syndrome ("GBS").
Respondent.	*	

Brett Slavicek, Brett L. Slavicek PC, Phoenix, AZ, for Petitioner.
Sarah Christina Duncan, U.S. Department of Justice, Washington, DC, for Respondent.

RULING ON ENTITLEMENT[1]

On April 9, 2020, Larry J. Parker ("Petitioner") filed a petition for compensation under the National Vaccine Injury Compensation Program ("Vaccine Act" or "the Program"), 42 U.S.C. § 300aa-10 et seq. (2018),[2] alleging that he suffered Guillain-Barré Syndrome ("GBS") as

[1] Because this Ruling contains a reasoned explanation for the action in this case, the undersigned is required to post it on the United States Court of Federal Claims' website and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). **This means the Ruling will be available to anyone with access to the Internet.** In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, the undersigned agrees that the identified material fits within this definition, the undersigned will redact such material from public access.

[2] The National Vaccine Injury Compensation Program is set forth in Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755, codified as amended, 42 U.S.C. §§ 300aa-10 to -34 (2018). All citations in this Ruling to individual sections of the Vaccine Act are to 42 U.S.C. § 300aa.

the result of a pneumococcal conjugate 13 valent ("Prevnar 13")[3] vaccination he received on April 4, 2017. Petition at Preamble, ¶ 24 (ECF No. 1); Amended ("Am.") Petition at Preamble, ¶ 24 (ECF No. 12). Respondent argued against compensation, "recommend[ing] that entitlement to compensation be denied." Respondent's Report ("Resp. Rept.") at 1 (ECF No. 31).

After carefully analyzing and weighing the evidence presented in accordance with the applicable legal standards, the undersigned finds that Petitioner has provided preponderant evidence that the Prevnar 13 vaccine he received caused his GBS, satisfying his burden of proof under Althen v. Secretary of Health & Human Services, 418 F.3d 1274, 1280 (Fed. Cir. 2005). Accordingly, Petitioner is entitled to compensation.

I. ISSUES TO BE DECIDED

The parties stipulate that Petitioner was 69 years old at the time of his Prevnar 13 vaccination on April 4, 2017, that the Prevnar 13 vaccine is recognized on the Vaccine Injury Table, and that Petitioner's Prevnar 13 vaccination was administered in the United States. Joint Submission, filed Jan. 17, 2023, at 1 (ECF No. 75).

Diagnosis is not at issue. The briefs submitted by both parties identify Petitioner's alleged vaccine-related injury as GBS. See Petitioner's Motion for Ruling on the Record ("Pet. Mot."), filed Jan. 12, 2023, at 16 (ECF No. 73) ("Petitioner was accurately diagnosed with GBS."); Resp. Response to Pet. Mot. ("Resp. Response"), filed Mar. 15, 2023, at 20 (ECF No. 76) ("There is no dispute that [P]etitioner's diagnosis of GBS is appropriate . . . ."); Pet. Reply in Support of Pet. Mot. ("Pet. Reply"), filed Apr. 4, 2023, at 1 (ECF No. 79) ("There is no dispute that Petitioner's diagnosis of GBS is accurate . . . .").

The parties dispute causation. Petitioner does not allege a Table injury, and thus, he must prove causation-in-fact by preponderant evidence. The parties dispute (1) whether the Prevnar 13 vaccine can cause GBS, (2) whether the Prevnar 13 vaccine Petitioner received on April 4, 2017 caused Petitioner's GBS, and (3) whether there is a medically appropriate temporal relationship between receipt of Petitioner's vaccination on April 4, 2017 and the onset of his GBS. Joint Submission at 1-2. Petitioner contends that he has provided preponderant evidence of the Althen criteria, and Respondent disagrees. Pet. Mot. at 16-17; Pet. Reply at 1-3; Resp. Response at 19-36.

II. BACKGROUND

A. Procedural History

Petitioner filed his petition on April 9, 2020, along with medical records, affidavits, an expert report from Dr. James Frey, and an amended petition in April and May 2020. Petition;

[3] The pleadings, filings, medical records, and expert reports use pneumococcal conjugate 13 valent, PCV13, PCV-13, and Prevnar 13 to refer to the vaccine at issue here. For consistency, the undersigned uses Prevnar 13 throughout the Ruling.

Am. Petition; Pet. Exhibits ("Exs.") 1-9.[4] This case was reassigned to the undersigned on July 31, 2020. Notice of Reassignment dated July 31, 2020 (ECF No. 18).

On April 23, 2021, Respondent filed his Rule 4(c) Report, recommending compensation be denied, along with expert reports from Dr. Brian C. Callaghan and Dr. You-Wen He. Resp. Rept.; Resp. Exs. A, C. On July 19, 2021, Petitioner filed updated medical records and an expert report from Dr. M. Eric Gershwin. Pet. Exs. 8g, 29. Respondent filed supplemental expert reports from Dr. He and Dr. Callaghan on December 1, 2021. Resp. Exs. E, G. Petitioner filed a supplemental expert report from Dr. Gershwin on May 31, 2022.[5] Pet. Ex. 34. On August 29, 2022, Respondent filed supplemental expert reports from Dr. He and Dr. Callaghan. Resp. Exs. H-I.

The undersigned held a Rule 5 conference on September 13, 2022. Rule 5 Order dated Sept. 13, 2022 (ECF No. 67). The undersigned preliminarily found that if this case were to go to hearing, she would likely find in favor of Petitioner on Althen prongs one, two, and three. Id. at 1-2. She encouraged the parties to informally resolve this matter if the parties were amenable. Id. at 2. However, on October 13, 2022, Respondent indicated he was not amenable to informal resolution. Resp. Status Rept., filed Oct. 13, 2022 (ECF No. 68). Petitioner filed updated medical records in November 2022. Pet. Exs. 6a, 8h, 64-66.

On November 14, 2022, Petitioner filed a joint status report, indicating that the parties preferred to resolve entitlement through a ruling on the record. Joint Status Rept. filed Nov. 14, 2022 (ECF No. 71). Thereafter, a briefing schedule was set and the parties filed their briefs from January to April 2023. Ruling on the Record Order dated Nov. 14, 2022 (ECF No. 72); Pet. Mot.; Resp. Response; Pet. Reply. Petitioner filed updated medical records on August 3, 2023. Pet. Ex. 8i.

This matter is now ripe for adjudication.

B. Medical Terminology

GBS is a rare illness that causes "acute flaccid symmetrical weakness of the limbs and areflexia usually reaching its peak within a month." Pet. Ex. 41 at 2.[6] Generally, GBS is "characterized by a rapidly progressive and self-limited weakness," which can "lead to

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[4] Petitioner filed many exhibits with repeating exhibit numbers during this time, and thus, these exhibits were not in compliance with the filing guidelines. Petitioner re-filed his exhibits in accordance with the filing guidelines in September 2020. See Pet. Exs. 1-28.

[5] Following the filing of this expert report, Respondent objected to Dr. Gershwin's reliance on improperly shared litigation materials in preparation of his expert report. Resp. Status Rept., filed June 6, 2022 (ECF No. 56). The parties conferred and the issue was resolved by July 26, 2022. Order dated July 26, 2022 (ECF No. 61).

[6] Anil K. Jasti et al., Guillain-Barré Syndrome: Causes, Immunopathogenic Mechanisms and Treatment, 12 Expert Rev. Clinical Immunology 1175 (2016).

respiratory failure[,] . . . paresthesias[,] [] pain, [and] other complications." Pet. Ex. 42 at 2.[7] The recovery process "can take months or years." Id. There are several types of GBS, with the subtype acute inflammatory demyelinating polyneuropathy ("AIDP") most common in North America. Id. at 4 tbl.1. Clinical features of AIDP include "multifocal patchy [d]emyelination," and some patients may have autonomic dysfunction, "including hypertension, hyperhidrosis, and blood pressure fluctuation." Pet. Ex. 41 at 8 tbl.4. Diagnosis is based on clinical history, physical examination, cerebrospinal fluid ("CSF") analysis, and electromyography ("EMG")/nerve conduction studies ("NCS"). Id. at 9.

GBS is thought to be an immune-mediated demyelinating illness, and it is usually treated with intravenous immunoglobulin ("IVIG") or plasma exchange. Pet. Ex. 41 at 2; Pet. Ex. 42 at 2, 11 tbl.2. The illness may be preceded by "respiratory or digestive infections." Pet. Ex. 41 at 3. It has also been associated with vaccines. See, e.g., id.; Resp. Ex. C, Tab 13 at 2.[8]

"Recent data suggest that [GBS] is primarily an antibody-mediated disorder . . . ." Resp. Ex. C, Tab 13 at 10. However, "[s]ince a wide range of viruses and bacterial agents" can trigger AIDP, "it has been difficult to find a common antigenic stimulus." Id. at 11. "Equally difficult has been the identification of specific antibody biomarkers in myelin." Id.

C. Summary of Medical Records[9]

1. Petitioner's Pre-Vaccination Medical History and Date of Vaccination

Petitioner was 69 years old at the time of the subject Prevnar 13 vaccination on April 4, 2017. Pet. Ex. 5 at 7. His pre-vaccination medical history was significant for anxiety, chronic low back pain, and renal insufficiency. Pet. Ex. 2k at 1-33; Pet. Ex. 5 at 7. He did not have any history of neurologic conditions. On April 4, 2017, Petitioner saw his primary care physician ("PCP"), Mark Lewis, M.D., for a routine medical exam and received a Prevnar 13 vaccination intramuscularly in his left deltoid. Pet. Ex. 5 at 7, 57. The medical records note that Petitioner was also supposed to receive a shingles vaccination; however, the shingles vaccination was not administered at that visit. Id. at 7, 57.

2. Petitioner's 2017 Medical History

On April 17, 2017, thirteen days after vaccination, Petitioner returned to Dr. Lewis

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[7] Paula Restrepo-Jiménez et al., The Immunotherapy of Guillain-Barré Syndrome, 19 Expert Op. on Biological Therapy 619 (2018).

[8] Peter D. Donofrio, Guillain-Barré Syndrome, 23 Continuum 1295 (2017).

[9] This summary of medical records is excerpted from Respondent's Response to Petitioner's Motion, as the undersigned finds it to be an accurate representation of the records. See Resp. Response at 2-15. The undersigned has deleted portions of the summary that are not relevant to Petitioner's diagnosis and treatment of GBS for the sake of brevity. In addition, citations to the record were changed if appropriate.

reporting four days of progressive, ascending numbness, tingling, and weakness. Pet. Ex. 5 at 12. Petitioner reported that the symptoms began in his right heel and progressed to include both of his legs, arms, tongue, and cheeks. Id. On physical examination, he had paresthesias bilaterally in his upper and lower extremities, weakness in both calves, and an abnormal gait and he was diagnosed with paresthesias. Id. at 13.

The next day, April 18, 2017, Petitioner presented to the emergency room ("ER") at Scottsdale Shea Hospital, with complaints of lower back pain, right leg weakness, and numbness. Pet. Ex. 9 at 5. Petitioner reported progressively worsening numbness and weakness in his legs, arms, mouth, tongue, and face, and that his calves and thighs gave out when standing. Id. On physical examination, Petitioner had normal reflexes and normal sensation. Id. at 7. Petitioner was admitted to the hospital for further workup. Id. at 9. While hospitalized, Petitioner underwent a cervical spine and brain magnetic resonance imaging ("MRI"), both of which were normal. Pet. Ex. 13 at 7-9. Petitioner also underwent a lumbar spine MRI which showed no significant stenosis, but some neural foraminal narrowing at the L4 level. Id. at 9-11. While in the hospital, Petitioner was given IV fluids and was seen by neurology and physical therapy ("PT"). Id. at 7, 49. PT recommended acute rehabilitation; however, it was not covered by Petitioner's insurance. Id. at 7. Petitioner was discharged on April 21, 2017, in stable condition to a nursing facility. Id. At discharge, his diagnoses included paresthesia, non-traumatic rhabdomyolysis, essential hypertension, mixed hyperlipidemia, and myalgia. Id. at 6. On April 21, 2017, Petitioner was admitted to Fountain View Village, a nursing facility, with diagnoses of paresthesia, weakness, low back pain, difficulty walking, rhabdomyolysis, myalgias, and depression. Pet. Ex. 4 at 8.

On April 22, 2017, emergency medical services ("EMS") was called to Fountain View Village upon reports that Petitioner was having extreme back pain due to a C4-5 herniation. Pet. Ex. 12 at 4. Petitioner was transported back to Scottsdale Shea Hospital for further care. Id. Upon admission at Scottsdale Shea Hospital, it was noted that Petitioner had continued myalgias, weakness, and focal paresthesias. Pet. Ex. 13 at 37. It was also noted that Petitioner had no change in symptoms since his last discharge, and no renal insufficiency. Id. at 37, 42. On physical examination, Petitioner's sensation was intact, and he was able to move all of his extremities. Id. at 39. Petitioner was admitted for non-traumatic rhabdomyolysis. Id. at 42.

Petitioner was transferred via EMS to the Mayo Clinic Hospital at his family's request on April 25, 2017. Pet. Ex. 12 at 12. Upon arrival at the Mayo Clinic Hospital ER, Petitioner reported a 10-day history of weakness, upper extremity paresthesias, and voice changes. Pet. Ex. 8a at 147. He reported falling and developing slurred speech and difficulty walking before his admission to Scottsdale Shea Hospital. Id. On physical examination, his deep tendon reflexes were absent in his patellar and Achilles tendons. Id. at 133. The impression was weakness and paresthesias of unclear etiology. Id. at 134.

Neurologist Erika Driver-Dunckley, M.D. saw Petitioner on April 26, 2017. Pet. Ex. 8a at 178. On physical examination, she noted that Petitioner had no weakness, but was diffusely areflexic, with diminished sensation in both ankles. Id. at 178-79. Her working diagnosis was lumbosacral radiculoplexopathy versus AIDP. Id. at 179. She noted that "[t]he patient's most debilitating symptom is that of severe pain in his bilateral lower extremities without evidence of

weakness, atrophy[,] or fasciculations on his examination. The patient has areflexia and some distal sensory findings." Id. The same day, Petitioner underwent an EMG which revealed findings "of a sensorimotor polyradiculoneuropathy with primarily demyelinating features." Pet. Ex. 8e at 664-66. Petitioner was discharged on May 5, 2017, with a diagnosis of GBS (AIDP), confirmed by EMG. Pet. Ex. 8a at 136. While admitted, Petitioner was treated with five rounds of IVIG and was started on gabapentin. Id. at 137. Upon discharge, Petitioner was transferred to inpatient rehabilitation at the Mayo Clinic. Id.

Petitioner was admitted to the Mayo Clinic's inpatient rehabilitation program from May 5, 2017 through May 19, 2017. See Pet. Ex. 8b at 259-63. At discharge, Petitioner's lower back pain was controlled, and he was ambulating with a cane. Id. at 263.

On May 26, 2017, Petitioner was seen by neurologist Benn Smith, M.D. in follow-up. Pet. Ex. 8e at 374. Dr. Smith's impression was AIDP. Id. at 375. Dr. Smith noted that Petitioner reached nadir in the hospital in late April, and he had been continuing with his recovery in rehabilitation. Id. Petitioner was approximately 75% recovered at that time. Id. By early June 2017, occupational therapy ("OT") noted that Petitioner was ambulating without an assistive device but was still unable to drive. Id. at 426, 434.

Neurologist Marie Grill, M.D. saw Petitioner on July 26, 2017. Pet. Ex. 8e at 361. Dr. Grill noted that Petitioner was ambulating independently and was able to lift 25 pounds, although he was still having lingering sensory issues. Id. at 362. Petitioner reported tingling in his arms and fingers, numbness in both feet and tingling around his mouth, and reported he was titrating off his gabapentin. Id. On physical examination, Petitioner had diminished sensation in his legs below the knees. Id. at 363. His gait was unstable, but he was able to walk unassisted. Id. Dr. Grill noted that Petitioner's sensory changes were likely due to decreasing the gabapentin. Id. at 364. Dr. Grill noted without further explanation, that Petitioner had an antecedent vaccination which "could have been the medium to prompt this immune reaction."[10] Id. On October 3, 2017, Petitioner returned to Dr. Grill for another follow-up. Id. at 367. At that time, it was noted that Petitioner had made significant improvements, but continued to have paresthesias in his upper extremities. Id. at 368. Petitioner was instructed to discontinue his gabapentin. Id.

3. Petitioner's 2018 Medical History

On January 5, 2018, Petitioner established care with family medicine practitioner Matthew Hummel, M.D. Pet. Ex. 6 at 6. At that visit, Petitioner reported continuing numbness in his legs and issues with hand dexterity and his short-term memory. Id. at 7. On physical examination, Petitioner had decreased fine motor movements in his hands and fingers and decreased sensation in his legs. Id. at 8.

Petitioner returned to Dr. Hummel on February 8, 2018 and reported hearing loss since his GBS, as well as balance issues, hip pain, tingling in his hands and toes, fine motor deficits in

[10] This quote in its entirety reads, "With respect to the causative agent regarding his [GBS], there was no antecedent illness noted though he did have a vaccination prior; thus, this certainly could have been the medium to prompt this immune reaction." Pet. Ex. 8e at 364.

his hands, and erectile dysfunction ("ED"). Pet. Ex. 6 at 10-12. On physical examination, Petitioner had poor hand dexterity and an irregular gait with abnormal sensation in the legs. Id. at 12. On March 2, 2018, Petitioner saw Dr. Hummel again, reporting hand motor deficits, short-term memory issues, hip pain, and numbness in his toes. Id. at 15-16. He reported that he had increased his walking and his mood had stabilized. Id. at 16-17. On examination, he had decreased hearing, bilateral hip tenderness, left sacroiliac joint tenderness, and abnormal sensation in his feet. Id. at 17.

On March 21, 2018, Petitioner began PT at 360 PT. Pet. Ex. 2 at 5. He was seen for bilateral bursitis in his hips, with an onset noted as approximately one year prior. Id. Petitioner reported that although his general condition had been improving, he developed an abdominal hernia and bilateral hip pain due to weight gain and weakness. Id.

Petitioner returned to Dr. Hummel with complaints of numbness in his toes, poor hand dexterity, and poor balance on March 30, 2018. Pet. Ex. 6 at 20-21.

On June 13, 2018, Petitioner presented to Nancy Koch, M.D., internal medicine, for a general examination. Pet. Ex. 8a at 9. Petitioner reported bilateral gluteal pain and memory loss since his April 2017 hospitalization. Id. at 9-10. On physical examination, he had no tenderness over his hips. Id. at 12. Dr. Koch noted that Petitioner's gait was normal at that time and Petitioner was no longer using gabapentin. Id. at 12-13. He was diagnosed with GBS with slowly resolving peripheral neuropathy and chronic renal insufficiency. Id. at 13-14.

Petitioner was discharged from 360 PT on June 29, 2018 after meeting his goals. Pet. Ex. 2 at 45.

On August 31, 2018, Petitioner was seen in the Mayo Clinic Hospital ER after a car accident. Pet. Ex. 8f at 481. He reported left neck and shoulder pain and denied any numbness or paresthesias. Id. On examination, he had intact sensation and normal gait. Id. at 482. After his car accident, Petitioner sought follow-up care with his PCP Dr. Hummel, orthopedist Vivek Agarwal, M.D., and a chiropractor Christi Chism, D.C. Pet. Ex. 6 at 47; Pet. Ex. 14 at 2; Pet. Ex. 15 at 3. Petitioner underwent a left shoulder MRI on September 11, 2018, which revealed a rotator cuff tear. Pet. Ex. 18 at 6. Petitioner also underwent a cervical spine MRI on September 21, 2018, which revealed severe bilateral foraminal stenosis at the C3-4 level, moderate stenosis at the C6-7 level, with possible impingement at the C4 level. Id. at 8. Petitioner ultimately sought care at a pain clinic due to left shoulder and neck pain from the car accident. Pet. Ex. 20 at 5. Petitioner also underwent PT in the fall of 2018 for his left shoulder and neck pain. Pet. Ex. 2 at 67.

Dr. Koch saw Petitioner on December 14, 2018 for complaints of recent onset of intermittent joint pains and aches after his car accident. Pet. Ex. 8f at 24. His pains had improved initially but worsened over the past month. Id. He was diagnosed with arthralgias. Id. at 27. On December 20, 2018, Dr. Koch ordered a rheumatology referral, noting that Petitioner's "symptoms can be from prior GBS but can also be a new autoimmune arthritis issue. Blood work shows he might have rheumatoid arthritis [("RA")]." Pet. Ex. 8a at 30.

4. Petitioner's 2019 Medical History

On January 21, 2019, Petitioner returned to see Dr. Koch. Pet. Ex. 8a at 34. He reported joint pains, pain in his knees, fingers, and hips, as well as worsening memory issues. Id. Dr. Koch diagnosed memory issues and suggested that Petitioner might be suffering from "mini strokes" over time. Id. at 37-38.

Petitioner presented to rheumatologist Fawad Aslam, M.D. to establish care for his joint and body pains on January 31, 2019. Pet. Ex. 8a at 43. Petitioner's pain was reportedly worse in the morning and evening. Id. at 44. Dr. Aslam noted that Petitioner had tingling and numbness since his GBS, but no other issues. Id. Petitioner was diagnosed with arthritis, but Dr. Aslam noted that his history and examination were not consistent with RA. Id. Dr. Aslam also noted that "[r]eactive arthritis has been described in association with GBS but it is rare[,] and he does not have the typical findings." Id. The same day, Petitioner underwent x-rays of his hand, foot, and hips, and the results were consistent with osteoarthritis. Id. at 120.

Dr. Aslam next saw Petitioner on February 25, 2019 for follow-up of his arthritis. Pet. Ex. 8a at 16. Dr. Aslam noted that Petitioner had a positive rheumatoid factor and cyclic citrullinated peptide ("CCP") antibody test. Id. Dr. Aslam ordered additional autoimmune testing. Id. Petitioner was diagnosed with clinically suspected RA and was prescribed hydroxychloroquine. Id.

On March 6, 2019, Petitioner returned to Dr. Hummel with complaints of hand swelling. Pet. Ex. 6 at 86-88. Dr. Hummel noted that Petitioner was seeing a rheumatologist for reactive arthritis. Id. at 88. Dr. Hummel's diagnoses included RA, inflammatory polyarthropathy, and GBS with deficits. Id. at 88-89. Dr. Hummel questioned whether Petitioner's RA was related to his GBS and noted Petitioner had likely reached a plateau with his GBS recovery. Id. at 89.

Petitioner returned to Dr. Aslam on June 3, 2019. Pet. Ex. 8f at 258. At that time, Petitioner was doing well on hydroxychloroquine, and reported only morning stiffness. Id. at 259. Dr. Aslam's diagnosis was RA, and he noted that Petitioner was "doing well." Id. at 260. He further noted that Petitioner's GBS was stable, but that Petitioner was "not a candidate for vaccinations due to his history of [GBS]." Id. at 261.

Petitioner was seen in a pain clinic for left shoulder pain from his car accident on July 25, 2019. Pet. Ex. 20 at 14. Petitioner reported residual numbness in his upper and lower extremities, which he attributed to his GBS. Id. On August 9, 2019, Petitioner was seen by Amon T. Ferry, M.D. at OrthoArizona – Arizona Sports Medicine Center for continued left shoulder pain after his car accident. Pet. Ex. 17 at 2. Dr. Ferry noted that Petitioner had a history of GBS, but felt he was about 85% resolved, with some residual neuropathy in his hands and feet. Id. On October 31, 2019, Petitioner returned to Dr. Hummel complaining of bilateral hip pain and left shoulder pain. Pet. Ex. 6 at 123, 125. On physical examination, he had limited range of motion in both hips and baseline lower extremity numbness. Id. at 125. He was again diagnosed with bursitis. Id.

On December 11, 2019, Petitioner saw Dr. Aslam in follow-up for his RA. Pet. Ex. 8g at 261. He continued to take hydroxychloroquine, which he was doing well on. Id. at 262. He reported more pain in the past two weeks, which he attributed to the weather. Id. Dr. Aslam noted that Petitioner had a car accident earlier that year in which he sustained an injury to his shoulder and had received an injection several months ago that was wearing off. Id. Petitioner reported his pain level as a four out of 10, up from his usual two or three out of 10. Id. He reported using over-the-counter Tylenol four or five times over the past two weeks, which provided some temporary relief. Id. Petitioner further reported he felt 50-60% better "since February." Id. The assessment was RA, indicating Petitioner was "[d]oing well," and "[s]table" GBS. Id. at 264. Petitioner was to return in six months. Id.

5. Petitioner's 2020-2022 Medical History[11]

Petitioner presented to establish care with Patress Persons, M.D. and for a routine follow-up on October 8, 2020. Pet. Ex. 8g at 141. Petitioner reported a history of GBS after an influenza vaccination. Id. He reported that "[h]e continue[d] to recover[,] but still [had] intermittent shocks of pain" in his arms and legs, neuropathy in his feet, and an unsteady gait. Id. at 142. Petitioner also noted a history of RA, which was symptomatically stable. Id. Dr. Persons noted that Petitioner had a history of depression and anxiety, diagnosed in 2014-2015 after his business went bankrupt, which was well-controlled with medication. Id. Petitioner reported fatigue and needing to use the restroom three to four times per night. Id. Dr. Persons felt that his nocturia was likely secondary to his benign prostatic hyperplasia ("BPH") and his fatigue was likely secondary to his nocturia. Id. at 147. He recommended continued follow up with an ophthalmologist and rheumatologist for his RA. Id.

Moving forward to 2021, on April 12, Petitioner saw Dr. Persons at the Mayo Clinic. Pet. Ex. 8h at 1; Pet. Ex. 8g at 29. Petitioner reported intermittent left shoulder pain due to a torn rotator cuff and stated he did not want surgery. Pet. Ex. 8h at 1. He reported he was using Voltaren gel daily, had not had any recent PT, and felt that his shoulder limited his quality of life at times. Id. He reported intermittent nerve pain in his lower extremities and gait instability, which he attributed to his prior GBS. Id. He felt that his gait had improved significantly since his hospitalization but had not returned entirely back to baseline. Id. Petitioner was assessed with hypertensive chronic kidney disease ("CKD"), stage three, depressive disorder, anxiety, left shoulder pain for which he was referred to PT, BPH with urinary tract symptoms for which he was on tamsulosin (Flomax), vitamin B12 deficiency and neuropathy for which he was prescribed cyanocobalamin (vitamin B12), bilateral dry eye syndrome, and skin cancer on his ear. Id. at 1-2.

On June 18, 2021, Petitioner returned to Dr. Persons. Pet. Ex. 8h at 3. Dr. Persons noted that Petitioner was diagnosed in 2019 with rheumatoid factor and anti-CCP positive, and antinuclear antibodies ("ANA"), human leukocyte antigen ("HLA"), and hepatitis C ("HCV") negative, RA. Id. Petitioner reported he was doing well with 15 to 20 minutes of morning stiffness and occasional right knee swelling. Id. On physical examination, he had no

[11] Portions of the summary that do not relate to GBS from 2020 to 2022 have been deleted for the sake of brevity.

neurological deficits. Id. at 3-4. Dr. Person's assessment was "[RA]/clinically suspect arthralgia," medication monitoring, and "[s]table" GBS. Id. at 4-5.

Petitioner saw Dr. Persons for a routine check up on April 12, 2022. Pet. Ex. 8h at 14. He reported he had "been doing well developing COVID19," and his symptoms had improved but he had a residual cough. Id. He reported that he had last seen optometry in January, and felt his vision was worsening. Id. He also reported difficulty hearing and that he did not wear his hearing aids. Id. Petitioner was assessed with hypertension and CKD, arthritis, depressive disorder, anxiety, vitamin B12 deficiency, fatigue, BPH, cough, allergic rhinitis, postnasal drip, skin lesions, blurred vision, bilateral hearing loss, obesity, and others. Id. at 15-16. These comorbidities were not associated with his history of GBS. Id. Dr. Persons wrote that Petitioner had a history of GBS, with stable, but not worsening neuropathy. Id. at 16. He did note that Petitioner reported significant ED ever since, for which he was referred to urology. Id.

On April 25, 2022, Petitioner was seen by rheumatology at the Mayo Clinic. Pet. Ex. 8h at 17. He reported he was doing very well and was not using over-the-counter pain medication more than once a month. Id. His GBS was noted as stable and he was instructed to continue hydroxychloroquine. Id. at 18.

Lastly, on September 22, 2022, Petitioner was seen by Dr. Hummel for a follow-up of his GBS. Pet. Ex. 6a at 15. Petitioner reported that he still had numbness in his feet, "shooting nerve discomfort in [his] legs," and poor balance but no recent falls. Id. at 18 (emphasis omitted). He reported "dexterity issues with [his] hands," and a slow reaction time with decreased concentration. Id. He attributed his ED to his GBS. Id. Dr. Hummel's assessment was that Petitioner's GBS had likely reached a plateau and continued him on vitamin B12 and recommended an exercise program. Id. at 19. He also advised against any vaccinations, including Covid-19 vaccination. Id. Dr. Hummel's assessment also included inflammatory polyarthropathy, bilateral hearing loss, ED, and weight gain, which he attributed to Petitioner's GBS. Id. at 19-20.

D. Affidavits[12]

1. Petitioner's Affidavit

Petitioner stated that he saw his PCP, Dr. Lewis, on April 4, 2017. Pet. Ex. 21 at ¶ 2. His wife, Kay Parker, attended this appointment with him. Id. At this appointment, Petitioner received a prescription for Lipitor[13] and a Prevnar 13 vaccination. Id. at ¶ 3; Pet. Ex. 27 at ¶ 2.

[12] While the undersigned has reviewed the affidavit of Dr. Matthew Hummel, it is not summarized because it primarily addresses issues related to damages, which are not the subject of this Ruling. See Pet. Ex. 25.

[13] Lipitor (atorvastatin calcium) "acts as an antihyperlipidemic by inhibiting cholesterol synthesis;" and is "used in treatment of hypercholesterolemia and other forms of dyslipidemia." Atorvastatin Calcium, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=4739 (last visited Dec. 4, 2023).

He fulfilled his Lipitor prescription immediately after the appointment and began taking it. Pet. Ex. 21 at ¶ 4. His PCP also directed Petitioner to get a shingles vaccine at CVS at this appointment, however, Petitioner did not receive this vaccine. Id. at ¶¶ 3, 5.

Petitioner averred that he "started experiencing weakness and numbness in [his] legs and feet about [eight] or [nine] days after the April 4, 2017 appointment," or "about April 13, 2017." Pet. Ex. 21 at ¶ 6; Pet. Ex. 27 at ¶ 3. He stated that his wife called the pharmacist, who indicated Petitioner should stop taking Lipitor. Pet. Ex. 21 at ¶ 6. Petitioner stopped taking Lipitor at that time and followed up with Dr. Lewis around April 17, 2017. Id. at ¶ 7. He averred that these symptoms he began experiencing on or about April 13, 2017 were later diagnosed to be GBS. Pet. Ex. 27 at ¶ 3.

2. Kay Parker's Affidavit

Kay Parker is the wife of Petitioner. Pet. Ex. 22 at ¶ 2. She attended Petitioner's April 4, 2017 appointment with his PCP. Id. at ¶ 4. At that appointment, he was administered the Prevnar 13 vaccine. Id. at ¶ 5. They fulfilled Petitioner's Lipitor prescription, and Petitioner began taking the prescription immediately. Id. at ¶ 6. Although Petitioner was directed by his PCP to receive a shingles vaccination, he did not. Id. at ¶¶ 5, 7.

She averred that Petitioner "started experiencing weakness and numbness in his legs and feet about [eight] or [nine] days after the April 4, 2017 appointment." Pet. Ex. 22 at ¶ 8. She called Petitioner's pharmacist, who instructed Petitioner to top taking Lipitor, which Petitioner did. Id. at ¶¶ 8-9. She and Petitioner followed up with Petitioner's PCP around April 17, 2017. Id. at ¶ 9.

E. Expert Reports[14]

1. Petitioner's Expert, Dr. James L. Frey[15]

a. Background and Qualifications

Dr. Frey received his M.D. from Duke University before completing an internship and neurology residency at University of Chicago Hospital & Clinics and Washington University Medical Center in St. Louis, Missouri. Pet. Ex. 67 at 1. He has worked as a neurologist at various practices and hospitals since 1977, in the neurology department at the Barrow Neurological Institute since 1978, and as a clinical professor of neurology at the University of Arizona School of Medicine since 2009. Id. at 1-2. He is board certified in neurology with a

[14] Although the undersigned has reviewed all the expert reports, this Ruling does not include every detail of the experts' opinions. Instead, the undersigned focuses on the experts' material opinions, as they relate to the relevant issue of causation.

[15] Petitioner filed an affidavit and one expert report from Dr. Frey. Pet. Exs. 23-24. Dr. Frey's affidavit verified that he was asked to provide expert opinions, and that his opinions were set forth in his expert report. Pet. Ex. 23 at 1.

specialty certification in vascular neurology and is licensed to practice in Illinois and Arizona. Id. Throughout his career, Dr. Frey has been a member of various professional societies and committees and a reviewer for journals. Id. at 2-3. He has also authored or co-authored 50 publications and has been a co-investigator or principal investigator on 60 research projects. Id. at 2-3, 24-34.

b. Opinion

Dr. Frey opined that "to a reasonable degree of medical certainty," Petitioner "developed [GBS] from the [Prevnar 13] vaccine administered on April 4, 2017." Pet. Ex. 24 at 12.

i. Althen Prong One

Dr. Frey began by explaining that GBS is an "autoimmune' inflammatory condition" caused when "the body's immune system aberrantly attacks its own tissue, the peripheral nervous system." Pet. Ex. 24 at 8. In the context of vaccines, he described the theory of "molecular mimicry" as the mechanism involved in this process of autoimmunity, explaining it occurs when there is similarity between membrane molecules in a vaccine and one's own tissue. Id. at 8-9. "When 'molecular mimicry' exists, the body's white blood cells cannot differentiate between the human tissues and the . . . vaccine that share[s] the membrane molecules and will attack both." Id. at 9. Dr. Frey referred to this as a "cross reaction" that involves molecular structures common to both the "target vaccine" and "those of the peripheral nervous system." Id. at 10.

In support of his opinion, Dr. Frey raised two points. First, he stated that medical literature[16] shows "a relationship between vaccination with [Prevnar 13] and the occurrence of GBS," although he acknowledged the "likelihood of co-occurrence is very low." Pet. Ex. 24 at 11. He explained, however, that a statistically "low" likelihood does not equate to "no" likelihood, and that in individual cases, an association "is best determined by the specific facts of the case." Id.

Second, Dr. Frey noted "GBS has been seen in persons infected with [] [*S*]*treptococcus pneumoniae* bacterium," or *S. pneumoniae*, which "implies that the white cells of the host (person with pneumonia) that are attacking the bacteria then cross react and attack the host's peripheral nervous system tissue based on molecular mimicry that exists between the [*S. pneumoniae*] bacterium and the host person's peripheral nervous system tissue." Pet. Ex. 24 at 11. Dr. Frey did not file or cite to any specific supportive medical literature.

ii. Althen Prongs Two and Three

Dr. Frey opined that Petitioner's clinical course, which he termed "neurological symptom evolution," was consistent with GBS, and that diagnosis was confirmed by neurological

[16] Dr. Frey's report did not include a reference list or citations to any medical literature; therefore, any references to literature are not supported.

examination and electrophysiological findings. Pet. Ex. 24 at 10. He also noted that the medical records and decision to treat Petitioner with IVIG also confirms the diagnosis of GBS. Id.

Next, Dr. Frey opined that there was no other infectious cause for Petitioner's GBS. Pet. Ex. 24 at 10. Petitioner had not recently had any upper respiratory of gastrointestinal illnesses and had no antecedent illness in the six-month period before vaccination. Id. Moreover, Petitioner had "no interceding infectious process, i.e., during the interval between vaccination and the onset of GBS." Id. Dr. Frey further explained that although Petitioner began taking Lipitor on the day that he received his Prevnar 13 vaccination, Lipitor is not known to cause GBS. Id.

Two years after he had GBS, Petitioner was diagnosed with RA. Pet. Ex. 24 at 11. Dr. Frey discussed the relevance of this fact, stating that RA is an "autoimmune inflammatory disease," and noting that Petitioner's development of a second autoimmune illness "strongly implies" that Petitioner had "a biological propensity" to develop autoimmune responses. Id. This suggested to Dr. Frey another reason that Petitioner's development of GBS after vaccination is "more assuredly interpretable as an endogenous autoimmune response, and not a chance occurrence." Id.

Regarding prong three, Dr. Frey opined that Petitioner's onset of GBS occurred within the six-to-eight-week time frame that GBS typically occurs after vaccination or infection. Pet. Ex. 24 at 9-10. He stated that "based on epidemiological observations, GBS is construed as a consequence of an infection or a vaccination if [it] occurs within [six]-[eight] weeks of onset of the infection or vaccination." Id. at 9.

2. Petitioner's Expert, Dr. M. Eric Gershwin[17]

a. Background and Qualifications

Dr. Gershwin is board certified in internal medicine, rheumatology, and allergy and clinical immunology. Pet. Ex. 29 at 1-2; Pet. Ex. 40 at 2. He completed his M.D. at Stanford University after which he completed an internship and residency in internal medicine at Tufts New England Medical Center and trained in rheumatology and immunology at the National Institutes of Health in Maryland. Pet. Ex. 29 at 1; Pet. Ex. 40 at 1-2. He currently works in the Division of Rheumatology, Allergy, and Clinical Immunology at the University of California Davis School of Medicine as Director of the Allergy-Clinical Immunology Program and as a professor. Pet. Ex. 29 at 2; Pet. Ex. 40 at 1. Dr. Gershwin has held various editor and reviewer positions on medical journals, and has authored or co-authored over 1,000 publications during his career. Pet. Ex. 29 at 2; Pet. Ex. 40 at 3-139. He "ha[s] seen and treated patients with a variety of issues in neuroimmunology and ha[s] published on several aspects of immune-mediated neuropathology," including GBS. Pet. Ex. 29 at 2.

[17] Petitioner filed two expert reports from Dr. Gershwin. Pet. Exs. 29, 34.

b. Opinion

Dr. Gershwin focused his opinions on the "concept of molecular mimicry and on the expected occurrence of rare events following vaccination." Pet. Ex. 29 at 2. He opined "were it not for the vaccination and more likely than not, [Petitioner] would not have developed GBS." Id. at 10.

i. Althen Prong One

Dr. Gershwin agreed with Petitioner's diagnosis of GBS and noted that the dispute between the parties focused on the theory of molecular mimicry. Pet. Ex. 29 at 2. In his first expert report, Dr. Gershwin discussed molecular mimicry, the difficulties in identifying homology, and host genetic susceptibility. See id. at 2-9. In his second report, Dr. Gershwin offered his opinions about how the Prevnar 13 vaccine can trigger GBS via molecular mimicry.[18] See Pet. Ex. 34 at 1-4.

At the outset of his second report, Dr. Gershwin explained that he was provided with a copy the Koller decision as well as a copy of the petitioner's expert, Dr. Lawrence Steinman's opinion in Koller.[19] Pet. Ex. 34 at 1; see Koller v. Sec'y of Health & Hum. Servs., No. 16-439V, 2021 WL 5027947 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). Dr. Gershwin explained that he "used [the Koller] materials as a springboard to review the literature cited and arrive at [his] own conclusions." Pet. Ex. 34 at 1. Dr. Gershwin reviewed the medical records, medical literature, and other filings, and formed his own opinions as to causation. Id.

[18] Although the undersigned reviewed both reports from Dr. Gershwin, only the opinions and material most relevant are discussed here, and these are from his second report. See generally Pet. Ex. 34. For a helpful overview of the adaptive immune system, relevant to Petitioner's theory, see Pet. Ex. 52 (Jose L. Sanchez-Trincado et al., Fundamentals and Methods for T- and B-Cell Epitope Prediction, 2017 J. Immunology Rsch. 1).

[19] In Koller, the special master found that the Prevnar 13 vaccine was the cause of the petitioner's GBS. Koller v. Sec'y of Health & Hum. Servs., No. 16-439V, 2021 WL 5027947, at *23 (Fed. Cl. Spec. Mstr. Oct. 8, 2021). Dr. Lawrence Steinman was petitioner's expert in that case. Id. at *2. Here, Dr. Gershwin acknowledged receipt and review of the Koller Ruling and the expert report authored by Dr. Steinman in Koller. Pet. Ex. 34 at 1. After the filing of this expert report from Dr. Gershwin, Respondent filed a status report noting and objecting to the improperly shared litigation materials and citing the applicable sections of the Vaccine Act and the relevant Vaccine Rules. Resp. Status Rept., filed June 6, 2022. A status conference was held to discuss the improperly shared materials, and the parties confirmed that the issue had been resolved. Order dated July 26, 2022.

1. Phosphoglycerol[20] in Serotypes 18C and 23F

The mechanism described by Dr. Gershwin involves homology between phosphoglycerol structure in two serotypes in the Prevnar 13 vaccine, present in the antigens of *S. pneumoniae* serotypes 18C and 23F. Pet. Ex. 34 at 1-4. The "cross-reactive mimic is phospholipid found within the myelin sheath." Id. at 4.

Based upon information obtained from the Prevnar 13 vaccine patent,[21] Dr. Gershwin explained that "a critical component of the immunogenicity[22] of the conjugated capsular polysaccharide antigens [in the vaccine] is the O-acetyl phosphate, otherwise known as the glycerol phosphate side chain[,] . . . a component of the saccharide epitope." Pet. Ex. 34 at 1. These saccharide components are "chemically bound to the phosphoglycerol." Id. "To improve the antigenicity . . . and [] augment the immune response following vaccination, the saccharide-phosphoglycerol complex is then chemically attached to CRM[,] . . . a nontoxic variant of diphtheria toxin[]." Id. at 2.

Dr. Gershwin cited Chang et al.[23] to support his opinion that the phosphoglycerol component is preserved and important for purposes of antigenicity during the process of making the vaccine. Pet. Ex. 34 at 1- 2 (citing Pet. Ex. 36). Chang et al. wrote "it is shown that glycerol-phosphate must be preserved for conserving adequate antigenicity of the 18C capsular polysaccharide." Pet. Ex. 36 at 1.

As explained by Dr. Gershwin, although gangliosides have historically been seen as the major target in GBS, "they are not the only potential targeted epitope." Pet. Ex. 34 at 2. He noted that "phospholipids are an essential component of myelin." Id. Citing Ho et al.,[24] Gilburd

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[20] Phospho- is a "prefix[] indicating the presence of phosphorus in a compound." Stedman's Medical Dictionary 1486 (28th ed. 2006). Glycerol is "[a] sweet viscous fluid obtained by the saponification of fats and fixed oils; used as a solvent, as a skin emollient, . . . and as a vehicle and sweetening agent." Stedman's at 820.

[21] U.S. patent 9,492,559 B2.

[22] Immunogenicity is defined as "the property that endows a substance with the capacity to provoke an immune response, or the degree to which a substance possesses this property." Immunogenicity, Dorland's Med. Dictionary Online, https://www.dorlandsonline.com/dorland/definition?id=24893 (last visited Dec. 4, 2023).

[23] Janoi Chang et al., Relevance of O-acetyl and Phosphoglycerol Groups for the Antigenicity of the *Streptococcus pneumoniae* Serotype 18C Capsular Polysaccharide, 30 Vaccine 7090 (2012).

[24] Peggy P. Ho et al., Identification of Naturally Occurring Fatty Acids of the Myelin Sheath That Resolve Neuroinflammation, 4 Sci. Translational Med. 1 (2012).

et al.,[25] and Chang et al., Dr. Gershwin showed that "the immune response against the two pneumococcal serotypes 18C and 23F[] have the potential to cross-react with epitopes in myelin and particularly against phospholipids attached to the phosphoglycerol component." Id. (citing Pet. Exs. 36, 38-39). Dr. Gershwin observed that Chang et al., showed that "the chemical conjugate between the saccharide components and phosphoglycerol remain[s] intact, in order for there to be adequate immune recognition or antigenicity, as least for serotype 18C." Id.

After discussing his opinion that phospholipids are essential components of myelin and a target for cross-reactivity, Dr. Gershwin next explained the importance of CRM_{197}. Pet. Ex. 34 at 2. "CRM_{197} is a variation of diphtheria toxin," which "differs from the toxic form of diphtheria by a single amino acid." Id. "When it is linked to the saccharides of the vaccine, it increases the likelihood of an effective host immune response following vaccination." Id.

Next, Dr. Gershwin cited a study by Bryson et al.,[26] which showed that "human antibodies [] react with a 23F serotype." Pet. Ex. 34 at 2 (citing Pet. Ex. 35 at 2). Dr. Gershwin concluded that "when humans mount an immune response against serotype 23F, the antibody epitope includes phosphoglycerol." Id.

In conclusion, Dr. Gershwin's theory is based on molecular mimicry. Pet. Ex. 34 at 4. He opined that here, "the mimic is the phosphoglycerol structure within at least two serotypes of Prevnar, 18C and 23F. The cross-reactive mimic is phospholipid found within the myelin sheath" of peripheral nerves, causing GBS. Id.

2. Case Reports

In addition to the literature discussed above, Dr. Gershwin referenced two case reports. The first was published by Ravishankar[27] about a patient who developed GBS about one month after a pneumococcal vaccination. Pet. Ex. 32 at 1. The second, from Conner et al.,[28] was about a 61-year-old male who developed symptoms of GBS within two weeks of receiving a Prevnar 13 vaccination. Pet. Ex. 31 at 1. CSF revealed elevated protein (79 mg/dL) and NCS confirmed severe sensorimotor demyelinating peripheral neuropathy. Id.

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[25] B. Gilburd et al., Autoantibodies to Phospholipids and Brain Extract in Patients with the Guillain-Barré Syndrome: Cross-Reactive or Pathogenic?, 16 Autoimmunity 57 (1993).

[26] Steve Bryson et al., Structures of Preferred Human IgV Genes-Based Protective Antibodies Identify How Conserved Residues Contact Diverse Antigens and Assign Source of Specificity to CDR3 Loop Variation, 196 J. Immunology 4723 (2016).

[27] Nidhi Ravishankar, Guillain-Barre Syndrome Following PCV Vaccine, 2 Clinics Surgery 1413 (2017).

[28] Chad Conner et al., 13-Valent Pneumococcal Conjugate Vaccine-Induced Guillain-Barré Syndrome, 158 Chest J. A59 (2020).

3. Response to Dr. He's Comments about Molecular Mimicry

Respondent's expert, Dr. He, opined that "the molecular mimicry theory fails fundamentally to explain how similarities can cause autoimmune diseases" and "the molecular [mimicry] theory would have predicted a 100% autoimmune disease occurrence rate in the general population upon vaccination, which cannot be true." Resp. Ex. E at 3.

Dr. Gershwin characterized Dr. He's opinion rejecting the mechanism of molecular mimicry as a "fatal flaw" that "lacks[s] scientific merit and [is] unsupported in the medical literature." Pet. Ex. 34 at 1. To illustrate the point, Dr. Gershwin searched and found "1,863 peer review citations . . . in 2022" using the key words "molecular mimicry and autoimmunity." Id. He also found "13,123 papers [] defining epitopes." Id. Dr. Gershwin also asserted that Dr. He's reliance on epidemiology fails to appreciate "the concept of individual variation." Id. Dr. Gershwin criticized these opinions, again, stating they lacked "scientific merit." Id.

3. Respondent's Expert, Dr. Brian C. Callaghan[29]

a. Background and Qualifications

Dr. Callaghan is "neuromuscular specialist with a primary interest in patients with neuropathy such as [GBS]." Resp. Ex. A at 1. After he obtained his M.D. at University of Pennsylvania Medical School, he completed an internship and neurology residency at University of Pennsylvania Medical Center and a neuromuscular fellowship at University of Michigan. Resp. Ex. B at 1. He is board certified in neurology and electrodiagnostic medicine. Id.; Resp. Ex. A at 1. Dr. Callaghan is a neurology professor at the University of Michigan and a Staff Physician at the Veterans Affairs ("VA") Ann Arbor Health System in Michigan. Resp. Ex. B at 1-2. He "ha[s] published more than 100 articles with most focusing on neuropathy including the appropriate diagnostic evaluation and treatment," and he "estimate[s] that [he] ha[s] seen more than 50 patients with GBS." Resp. Ex. A at 1; see also Resp. Ex. B at 10-17.

b. Opinion

Dr. Callaghan agreed that Petitioner had GBS but disagreed as to the cause of his GBS, opining there was "no convincing, reliable evidence" that the Prevnar 13 vaccine can cause GBS. Resp. Ex. A at 4; see also Resp. Ex. G at 2; Resp. Ex. H at 2.

i. Althen Prong One

Dr. Callaghan first opined that "there [was] no convincing evidence supporting a likely association with Prevnar vaccination and GBS." Resp. Ex. A at 3. While he acknowledged that

[29] Respondent filed three expert reports from Dr. Callaghan. Resp. Ex. A, G-H.

there was an association between some influenza vaccinations and GBS,[30] he disagreed that there was any such association between the Prevnar 13 vaccination and GBS. Id. He cited two peer reviewed articles in support of his opinion.

Haber et al.[31] studied Vaccine Adverse Event Reporting System ("VAERS") reports in adults from June 2012 through December 2015 related to the Prevnar 13 vaccine. Resp. Ex. A, Tab 2 at 1. During that time, there were 2,976 total reports. Id. Most of the reports related to injection site adverse events (injection site pain, redness, and swelling). Id. at 2, 3 tbl.1. There were 11 cases of GBS reported following the Prevnar 13 vaccination, and in 10 of those, the Prevnar 13 vaccine was the only vaccine administered. Id. at 4. One patient also received an influenza vaccine. Id. The authors concluded that their "data mining analysis noted no disproportionate reporting for GBS." Id. at 5. The second article was from Martín Arias et al.,[32] which addressed the influenza vaccine, not the Prevnar 13 vaccine. See Resp. Ex. A, Tab 3. The authors reported "a small but statistically significant increase in the risk of developing GBS associated with influenza vaccines—seasonal or pandemic; in the case of the seasonal vaccines, it should be considered as marginally statistically significant." Id. at 3.

Next, Dr. Callaghan opined that Dr. Frey did not provide reliable evidence to show that molecular mimicry was a "potential mechanism" for vaccinations in general, or the Prevnar 13 vaccine specifically, to cause GBS. Resp. Ex. A at 3. The Martín Arias et al. article cited by Dr. Callaghan stated

> GBS is believed to be an immune disorder resulting from the generation of autoimmune antibodies that cross-react with epitopes on peripheral nerves, leading to nerve damage; active immunization by a vaccine, in turn, does stimulate the immune system to produce antigen-specific immunity. Thus, immune stimulation induced by vaccination could theoretically result in GBS; several mechanisms have been accordingly proposed: (i) the epitopes of a vaccine could initiate the development of antibodies . . . that could cross-react with epitopes on myelin . . . .

Resp. Ex. A, Tab 3 at 3 (internal citations omitted). The mechanism described by Martín Arias et al. is molecular mimicry, discussed by both Dr. Frey and Dr. Gershwin.

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[30] See, e.g., Resp. Ex. A, Tab 1 (James S. Marks & Thomas J. Halpin, Guillain-Barré Syndrome in Recipients of A/New Jersey Influenza Vaccine, 243 JAMA 2490 (1980)).

[31] Penina Haber et al., Post-Licensure Surveillance of 13-Valent Pneumococcal Conjugate Vaccine (PCV13) in Adults Aged ≥19 Years Old in the United States, Vaccine Adverse Event Reporting System (VAERS), June 1, 2012–December 31, 2015, 34 Vaccine 6330 (2016). This article was also cited by Respondent's expert, Dr. He. Resp. Ex. C, Tab 8.

[32] L.H. Martín Arias et al., Guillain-Barré Syndrome and Influenza Vaccines: A-Meta Analysis, 33 Vaccine 3773 (2015).

Dr. Callaghan cited the "traditional criteria for molecular mimicry," which Dr. Gershwin also cited:

> 1) similarity between a host epitope and an epitope of a microorganism or environmental agent, 2) detection of antibodies or T-cells that cross-react with both epitopes in patients with [autoimmune disease], 3) epidemiological link between exposure to the environmental agent or microbe and development of [autoimmune disease], and 4) reproducibility of autoimmunity in an animal model following sensitization with the appropriate epitopes either following infection with the microbe or exposure to the environmental agent.

Resp. Ex. H at 1 (internal quotations omitted); see Pet. Ex. 29 at 6.

Dr. Callaghan opined that these criteria have not been met, and "therefore, molecular mimicry has not been established as a likely mechanism for Prevnar [13] vaccination to lead to GBS." Resp. Ex. H at 1.

Lastly, regarding Petitioner's experts' reference to the Ravishankar case report, Dr. Callaghan opined that a single case report is insufficient to establish causation. Resp. Ex. H at 1-2.

ii. Althen Prong Two and Althen Prong Three

Dr. Callaghan agreed that Petitioner had GBS. Resp. Ex. H at 2. He did not opine that there was an alternative cause of Petitioner's GBS. See Resp. Exs. A, G-H. And he agreed that "the evidence [] suggests a proximate temporal relationship between [Petitioner's] Prevnar [13] vaccination and GBS," but opined that a temporal relationship alone is "insufficient to prove causation." Resp. Ex. H at 1-2.

4. Respondent's Expert, Dr. You-Wen He[33]

a. Background and Qualifications

Dr. He is currently a Professor of Immunology at the Department of Immunology at Duke University Medical Center. Resp. Ex. C at 1. Dr. He received his M.D. from the Fourth Military Medical University in Xian, China, and his Ph.D. in Microbiology and Immunology from the University of Miami School of Medicine in Miami, Florida. Resp. Ex. F at 1. "[His] research areas include both innate and adaptive immunity against viral and bacterial infections[,] as well as tumors." Resp. Ex. C at 1. He has conducted research on human immune responses to viral infections and is currently a Co-Principal Investigator for clinical trials focusing on cancer immunotherapy. Id. Dr. He has reviewed National Institutes of Health studies, serves on editorial boards, and has authored or co-authored over 130 publications. Id. at 1-2; Resp. Ex. F at 1-3, 8-17.

[33] Respondent filed three expert reports from Dr. He. Resp. Exs. C, E, I.

b. Opinion

Dr. He does not dispute Petitioner's diagnosis of GBS. In his summary of the medical records, Dr. He acknowledged that "[Petitioner] received [Prevnar 13] vaccine on April 4, 2017 and approximately 10 days later, he developed symptoms that were consistent with GBS." Resp. Ex. C at 3. However, Dr. He does dispute causation, opining "there is no evidence to associate [Prevnar 13] vaccination to GBS development." Id. at 4. Thus, he concluded, "to a reasonable degree of medical certainty[,] that there is no reliable evidence to support the experts' theory that [Prevnar 13] vaccine may have caused [Petitioner's] GBS." Id. at 12.

i. Althen Prong One

In his first expert report, Dr. He opined that there is no evidence to associate the Prevnar 13 vaccine with GBS. Resp. Ex. C at 3-4. He acknowledged that the vaccine contains 13 serotypes of pneumococcus, including 18C and 23F, conjugated to the diphtheria carrier protein CRM_{197}, but denied that there was any "causative link" between the vaccine and GBS. Id.

According to Dr. He, "two essential criteria" must be fulfilled in order to reach conclusions about the cause of a disease: (1) epidemiologic evidence and (2) mechanistic evidence. Resp. Ex. C at 7. Dr. He cited the 2012 Institute of Medicine ("IOM") report and several articles to support his position that there is no epidemiological support for a causal association here. Id. He stated,

> [a] potential causative link . . . documenting GBS and various vaccinations[] has been thoroughly analyzed by the [IOM] of the National Academy of Sciences and the Agency for Healthcare Research and Quality and further updated in a recent study. These authoritative [systematic] studies thoroughly analyze both epidemiologic and mechanistic evidence for disease causality. However, none of these [] studies even listed [Prevnar 13] as a potential cause for GBS development due to a complete lack of evidence.

Id. at 3 (citing Resp. Ex. C, Tab 4;[34] Resp. Ex. C, Tab 5;[35] Resp. Ex. C, Tab 6;[36] Resp. Ex. C, Tab 7).[37]

Dr. He's reliance on the 2012 IOM report and the other papers appears to be misplaced. As explained by Maglione et al., "[t]he IOM did not study the safety of [Prevnar 13]." Resp. Ex. C, Tab 5 at 8. And in the initial summary of the IOM report, the editors confirmed the vaccines reviewed and studied, and this list did not include any pneumococcal vaccine. See Court Ex. 1 at 2.[38] Thus, it was erroneous for Dr. He to opine that the IOM "thoroughly analyze[d]" Prevnar 13 as a potential cause of GBS. Resp. Ex. C at 3.

The next article cited by Dr. He for the proposition that the Prevnar 13 vaccine has been "thoroughly analyze[d]" was authored by Maglione et al. Resp. Ex. C, Tab 5. The purpose of the study was to update the IOM report by summarizing newer literature and expand the scope of vaccines reviewed. Id. at 1. The study excluded the first pneumococcal conjugate vaccine administered in the United States (pneumococcal conjugate vaccine 7),[39] but did include Prevnar 13. Id. at 2. The authors performed a systematic review of literature from one year before the IOM report was published (2011) through August 2013. Id. Relevant to Prevnar 13, one paper

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[34] Inst. of Med., Evaluating Biological Mechanisms of Adverse Events, in Adverse Effects of Vaccines: Evidence and Causality 57, 70-71 (Kathleen Stratton et al. eds., 2012). Only two pages from this chapter were filed.

[35] Margaret A. Maglione et al., Safety of Vaccines Used for Routine Immunization of US Children: A Systematic Review, 134 Pediatrics 325 (2014).

[36] Agency for Healthcare Rsch. & Quality, Safety of Vaccines Used for Routine Immunization in the United States (Apr. 6, 2020), https://effectivehealthcare.ahrq.gov/products/safety-vaccines/protocol. The 2014 version was cited in Dr. He's report; however, the 2020 research protocol version was filed in the case.

[37] Matthew Z. Dudley et al., The Safety of Vaccine Safety Science: Systematic Reviews of the Evidence, 20 Lancet Infectious Diseases e80 (2020).

[38] Inst. of Med., Summary, in Adverse Effects of Vaccines: Evidence and Causality, supra note 34 at 1-2. Dr. He filed only an excerpt of the IOM report that describes the mechanism of molecular mimicry. See Resp. Ex. C, Tab 4. Court Exhibit 1 is an additional excerpt from the summary of the report, which identifies the vaccines studied.

[39] Pneumococcal conjugate vaccine 7 was licensed in 2000 and Prevnar 13 was licensed in 2010. About Pneumococcal Vaccines, Ctrs. for Disease Control & Prevention, https://www.cdc.gov/vaccines/vpd/pneumo/hcp/about-vaccine.html (last reviewed Sept. 21, 2023). At the time of the Maglione et al. study, pneumococcal conjugate vaccine 7 was no longer administered, and thus, was excluded from the study. Resp. Ex. C, Tab 5 at 2.

was cited,[40] which reported on the risk of febrile seizures following the trivalent inactivated influenza vaccination. Id. at 8. The authors noted that the Prevnar 13 vaccination was also associated with an increased risk of febrile seizures in children less than five years of age, especially when administered at the same time as the influenza vaccination. Id. Since the referenced article was not filed, the methodology, scope of adverse events studied, and outcomes are not available. Thus, it is not clear whether the adverse event of GBS was studied. Based on the title of the article, and the brief excerpt in Maglione et al., it appears that the study was limited to children under the age of five and it does not appear that the Prevnar 13 vaccine was the focus of the study. Id.

Next, Dr. He referenced a review article by Dudley et al., published in 2020. Resp. Ex. C, Tab 7. Like Maglione et al., the purpose of Dudley et al. was to provide an update to the 2012 IOM report about "possible causal associations of adverse events" following vaccination. Id. at 1. GBS was identified as an adverse event, and the authors concluded that the "[i]nfluenza vaccine can cause [GBS] very rarely in adults." Id. at 4 tbl.1. However, the authors did not identify the Prevnar 13 vaccine as one of the vaccines examined in relation to GBS. See id.

Dr. He also cited Haber et al., where the authors examined VAERS reports and identified 11 cases of GBS after Prevnar 13 vaccination. Resp. Ex. C at 3-4 (citing Resp. Ex. A, Tab 2 at 1, 4). The authors concluded that there was no disproportionate reporting for GBS. Id. at 4. Dr. He opined that VAERS data is not "reliable" evidence to support a causal relationship since it is a "passive reporting system," the diagnoses in the reports are not confirmed, and alternative causes are not known. Id. at 4.

In addition to offering opinions about the safety studies and medical literature, Dr. He addressed Dr. Frey's opinions about molecular mimicry. Resp. Ex. C at 4-12. Dr. He agreed that molecular mimicry is based on the hypothesis that "shared antigenic epitope similarity between infectious pathogens or vaccines and human proteins renders cross-reactivities by host adaptive immunity" and as a result of this cross-reactivity, "cellular and humoral immune responses induced by infection and vaccination attack self-tissues." Id. at 5. Although he recognized the theory of molecular mimicry, Dr. He opined it is an "old theory that has been strongly challenged by recent scientific evidence." Id. Based on the findings reported by Kanduc et al.,[41] Dr. He asserted that "viral and human proteomes have massive peptide sharing," which would cause "a 100% autoimmune disease rate in the general population after either infection or vaccination" if molecular mimicry was a relevant causal mechanism. Id. (citing Resp. Ex. C, Tab 9). Dr. He concluded that "[m]ere sequence similarity cannot be used to [conclude] that vaccines cause autoimmune diseases." Id.

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[40] Alison Tse et al, Signal Identification and Evaluation for Risk of Febrile Seizures in Children Following Trivalent Inactivated Influenza Vaccine in the Vaccine Safety Datalink Project, 2010-2011, 30 Vaccine 2024 (2012), https://pubmed.ncbi.nlm.nih.gov/22361304/. This paper was not filed in the present case.

[41] Darja Kanduc et al., Massive Peptide Sharing Between Viral and Human Proteomes, 29 Peptides 1755 (2008).

Although Dr. He challenged the role of molecular mimicry as a causal theory for post-vaccination GBS, he cited a number of articles (published after Kanduc et al.) that discuss molecular mimicry and its role in the pathogenesis of GBS. For example, a paper published in Lancet in 2016 from Willison et al.[42] recognized that GBS is "usually preceded by infection or other immune stimulation that induces an aberrant autoimmune response targeting peripheral nerves and their spinal roots. Molecular mimicry between microbial and nerve antigens is clearly a major driving force behind the development of the disorder, at least in the case of *Campylobacter jejuni* infection." Resp. Ex. C, Tab 2 at 1. And in 2015, Martín Arias et al. stated that "immune stimulation induced by vaccination could theoretically result in GBS." Resp. Ex. A, Tab 3 at 3. The authors discussed the mechanism of molecular mimicry—vaccine epitopes could initiate antibodies that cross-react with epitopes on myelin. Id.

Dr. He then turned to Dr. Frey's opinion that *S. pneumoniae* infection, which the Prevnar 13 vaccination provides immunity against, is a potential cause of GBS. Resp. Ex. C at 6-7, 9, 11. Dr. He responded by stating there is "no scientific evidence" that this infection can cause GBS, and thus, he opined that Dr. Frey's opinion is "complete speculation." Id. at 11.

In his second expert report, Dr. He addressed Dr. Gershwin's opinions about molecular mimicry, specifically "the existence of secondary and tertiary structures of antigenic epitopes." Resp. Ex. E at 3. Dr. He "fully agree[d]" with Dr. Gershwin's discussion on these points, but disagreed that molecular mimicry explains the cause of autoimmune illnesses. Id. Dr. He also disagreed with Dr. Gershwin that epidemiology fails to detect rare events. Id. at 5. Lastly, he criticized the case reports cited by Dr. Gershwin, opining they failed to support a causal link between Prevnar 13 vaccination and GBS. Id. at 6. Regarding the case report from Ravishankar, Dr. He criticized the long temporal association, the publication of the case in two different journals, and the low quality of the publications. Id.

In his third expert report, Dr. He responded to Dr. Gershwin's hypothesis based on the glycerol phosphate side chains in serotypes 18C and 23F of the vaccine. Resp. Ex. I at 2-6. Dr. He offered three criticisms. First, he stated that there was no proof that Petitioner had "anti-glycerol phosphate side chain antibodies" after Prevnar 13 vaccination, which is addressed in more detail below. Id. at 4. Second, even if such antibodies were present after vaccination, "it is unknown whether these antibodies cross-react to phospholipids attached to myelin." Id. And third, "even if there were cross-reactivities to myelin protein, it is unknown whether these antibodies play any pathological role[] in GBS." Id. Dr. He acknowledged that Gilburd et al. showed that anti-phospholipids have been detected in GBS patients. Id. at 5 (citing Pet. Ex. 39). But he emphasized the conclusion in Gilburd et al. stated "these autoantibodies are probably produced as a result of the myelin damage rather than [the] cause [of] the demyelination." Id. (quoting Pet. Ex. 39 at 1).

Dr. He did not take issue with Dr. Gershwin's description of foundational data showing the glycerol phosphate side chains were present in serotypes 18C and 23F of the Prevnar 13

[42] Hugh J. Willison et al., Guillain-Barré Syndrome, 388 Lancet 717 (2016). This article was also filed as Resp. Ex. E, Tab 1.

vaccine or that they could illicit an immune response. Resp. Ex. I at 4-5. He cited Chang et al. and noted the confirmation of the glycerol phosphate and O-acetyl group on serotype 18C. Id. (citing Pet. Ex. 36). And he referenced Yu et al.,[43] which described the serotype 23F. Id. at 5 (citing Pet. Ex. 37). However, Dr. He disagreed there was evidence that the "[Prevnar 13] vaccination produces . . . antibodies that [] cross react with myelin protein," or play any causal role in GBS. Id. at 4.

ii. Althen Prongs Two and Three

Dr. He agreed that "[Petitioner] received the [Prevnar 13] vaccine on April 4, 2017 and approximately 10 days later, [] developed symptoms that were consistent with GBS." Resp. Ex. C at 3. Dr. He agreed with the diagnosis of GBS. See id.

Regarding the mechanism proposed by Dr. Gershwin, Dr. He stated that that there was no proof that Petitioner had "anti-glycerol phosphate side chain antibodies upon [Prevnar 13] vaccination," as would be expected given the causal mechanism proposed by Dr. Gershwin.[44] Resp. Ex. E at 4-5; see Pet. Ex. 39 (reporting serum autoantibodies to various phospholipids in patients with GBS).

Dr. He did not identify any alternative antecedent or intercurrent infectious cause for Petitioner's GBS. Resp. Ex. C at 8. Instead, he noted that "a third of GBS cases have no known infection." Id. The medical records show that Petitioner and his initial treating physicians were concerned that his symptoms were caused by Lipitor. Id. Although Lipitor was initiated on the same day as Petitioner's Prevnar 13 vaccine, Dr. He agreed that "there is no literature to support . . . the potential for Lipitor [] to cause GBS." Id. In summary, Dr. He did not opine as to any alterative cause, infectious or medication related, for Petitioner's GBS.

Regarding timing, Dr. He agreed that "a temporal relationship exists for [Petitioner's] GBS development." Resp. Ex. C at 8. However, he attributed the temporal relationship to coincidence and not vaccination. Id.

III. DISCUSSION

A. Standards for Adjudication

The Vaccine Act was established to compensate vaccine-related injuries and deaths. § 10(a). "Congress designed the Vaccine Program to supplement the state law civil tort system as a simple, fair and expeditious means for compensating vaccine-related injured persons. The

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[43] Kang Yu et al., Synthesis of the Biological Repeating Unit of *Streptococcus pneumoniae* Serotype 23F Capsular Polysaccharide, 14 Organic & Biomolecular Chemistry 11462 (2016).

[44] There is no evidence in the medical records that tests were performed to detect the presence of these autoantibodies in Petitioner. The availability of such testing was not discussed by the experts.

Program was established to award 'vaccine-injured persons quickly, easily, and with certainty and generosity.'" Rooks v. Sec'y of Health & Hum. Servs., 35 Fed. Cl. 1, 7 (1996) (quoting H.R. Rep. No. 908 at 3, reprinted in 1986 U.S.C.C.A.N. at 6287, 6344).

Petitioner's burden of proof is by a preponderance of the evidence. § 13(a)(1). The preponderance standard requires a petitioner to demonstrate that it is more likely than not that the vaccine at issue caused the injury. Moberly v. Sec'y of Health & Hum. Servs., 592 F.3d 1315, 1322 n.2 (Fed. Cir. 2010). Proof of medical certainty is not required. Bunting v. Sec'y of Health & Hum. Servs., 931 F.2d 867, 873 (Fed. Cir. 1991). Petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano v. Sec'y of Health & Hum. Servs., 440 F.3d 1317, 1325 (Fed. Cir. 2006). Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

In particular, a petitioner must prove that the vaccine was "not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface v. Sec'y of Health & Hum. Servs., 165 F.3d 1344, 1352-53 (Fed. Cir. 1999)); see also Pafford v. Sec'y of Health & Hum. Servs., 451 F.3d 1352, 1355 (Fed. Cir. 2006). The received vaccine, however, need not be the predominant cause of the injury. Shyface, 165 F.3d at 1351. A petitioner who satisfies this burden is entitled to compensation unless Respondent can prove, by a preponderance of the evidence, that the vaccinee's injury is "due to factors unrelated to the administration of the vaccine." § 13(a)(1)(B). However, if a petitioner fails to establish a prima facie case, the burden does not shift. Bradley v. Sec'y of Health & Hum. Servs., 991 F.2d 1570, 1575 (Fed. Cir. 1993).

"Regardless of whether the burden ever shifts to the [R]espondent, the special master may consider the evidence presented by the [R]espondent in determining whether the [P]etitioner has established a prima facie case." Flores v. Sec'y of Health & Hum. Servs., 115 Fed. Cl. 157, 162-63 (2014); see also Stone v. Sec'y of Health & Hum. Servs., 676 F.3d 1373, 1379 (Fed. Cir. 2012) ("[E]vidence of other possible sources of injury can be relevant not only to the 'factors unrelated' defense, but also to whether prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question."); de Bazan v. Sec'y of Health & Hum. Servs., 539 F.3d 1347, 1353 (Fed. Cir. 2008) ("The government, like any defendant, is permitted to offer evidence to demonstrate the inadequacy of the [P]etitioner's evidence on a requisite element of the [P]etitioner's case-in-chief."); Pafford, 451 F.3d at 1358-59 ("[T]he presence of multiple potential causative agents makes it difficult to attribute 'but for' causation to the vaccination. . . . [T]he Special Master properly introduced the presence of the other unrelated contemporaneous events as just as likely to have been the triggering event as the vaccinations.").

B. Factual Issues

Petitioner must prove, by a preponderance of the evidence, the factual circumstances surrounding his claim. § 13(a)(1)(A). To resolve factual issues, the special master must weigh the evidence presented, which may include contemporaneous medical records and testimony. See Burns v. Sec'y of Health & Hum. Servs., 3 F.3d 415, 417 (Fed. Cir. 1993) (explaining that a

special master must decide what weight to give evidence including oral testimony and contemporaneous medical records).

Contemporaneous medical records, "in general, warrant consideration as trustworthy evidence." Cucuras v. Sec'y of Health & Hum. Servs., 993 F.2d 1525, 1528 (Fed. Cir. 1993). But see Kirby v. Sec'y of Health & Hum. Servs., 997 F.3d 1378, 1382 (Fed. Cir. 2021) (rejecting the presumption that "medical records are accurate and complete as to all the patient's physical conditions"); Shapiro v. Sec'y of Health & Hum. Servs., 101 Fed. Cl. 532, 538 (2011) ("[T]he absence of a reference to a condition or circumstance is much less significant than a reference which negates the existence of the condition or circumstance." (quoting Murphy v. Sec'y of Health & Hum. Servs., 57 Cl. Ct. 726, 733 (1991), aff'd per curiam, 968 F.2d 1226 (Fed. Cir. 1992))), recons. den'd after remand, 105 Fed. Cl. 353 (2012), aff'd mem., 503 F. App'x 952 (Fed. Cir. 2013). The weight afforded to contemporaneous records is due to the fact that they "contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium." Id. To overcome the presumptive accuracy of medical records, a petitioner may present testimony which is "consistent, clear, cogent, and compelling." Sanchez v. Sec'y of Health & Hum. Servs., No. 11-685V, 2013 WL 1880825, at *3 (Fed. Cl. Spec. Mstr. Apr. 10, 2013) (citing Blutstein v. Sec'y of Health & Hum. Servs., No. 90-2808V, 1998 WL 408611, at *5 (Fed. Cl. Spec. Mstr. June 30, 1998)), mot. for rev. denied, 142 Fed. Cl. 247 (2019), vacated on other grounds & remanded, 809 F. App'x 843 (Fed Cir. 2020).

There are situations in which compelling testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. Campbell v. Sec'y of Health & Hum. Servs., 69 Fed. Cl. 775, 779 (2006) ("[L]ike any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking."); Lowrie v. Sec'y of Health & Hum. Servs., No. 03-1585V, 2005 WL 6117475, at *19 (Fed. Cl. Spec. Mstr. Dec. 12, 2005) ("[W]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent." (quoting Murphy, 57 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. Andreu v. Sec'y of Health & Hum. Servs., 569 F.3d 1367, 1379 (Fed. Cir. 2009); Bradley, 991 F.2d at 1575.

Despite the weight afforded to medical records, special masters are not bound rigidly by those records in determining onset of a petitioner's symptoms. Valenzuela v. Sec'y of Health & Hum. Servs., No. 90-1002V, 1991 WL 182241, at *3 (Fed. Cl. Spec. Mstr. Aug. 30, 1991); see also Eng v. Sec'y of Health & Hum. Servs., No. 90-1754V, 1994 WL 67704, at *3 (Fed. Cl. Spec. Mstr. Feb. 18, 1994) (Section 13(b)(2) "must be construed so as to give effect also to § 13(b)(1) which directs the special master or court to consider the medical records (reports, diagnosis, conclusions, medical judgment, test reports, etc.), but does not require the special master or court to be bound by them").

C. Causation

To receive compensation through the Program, Petitioner must prove either (1) that he suffered a "Table Injury"—i.e., an injury listed on the Vaccine Injury Table—corresponding to a vaccine that he received, or (2) that he suffered an injury that was actually caused by a vaccination. See §§ 11(c)(1), 13(a)(1)(A); Capizzano, 440 F.3d at 1319-20. Petitioner must show that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." Moberly, 592 F.3d at 1321 (quoting Shyface, 165 F.3d at 1352-53).

Because Petitioner does not allege he suffered a Table Injury, he must prove a vaccine he received caused his injury. To do so, Petitioner must establish, by preponderant evidence: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." Althen, 418 F.3d at 1278.

The causation theory must relate to the injury alleged. Petitioner must provide a sound and reliable medical or scientific explanation that pertains specifically to this case, although the explanation need only be "legally probable, not medically or scientifically certain." Knudsen v. Sec'y of Health & Hum. Servs., 35 F.3d 543, 548-49 (Fed. Cir. 1994). Petitioner cannot establish entitlement to compensation based solely on her assertions; rather, a vaccine claim must be supported either by medical records or by the opinion of a medical doctor. § 13(a)(1). In determining whether Petitioner is entitled to compensation, the special master shall consider all material in the record, including "any . . . conclusion, [or] medical judgment . . . which is contained in the record regarding . . . causation." § 13(b)(1)(A). The special master must weigh the submitted evidence and the testimony of the parties' proffered experts and rule in Petitioner's favor when the evidence weighs in his favor. See Moberly, 592 F.3d at 1325-26 ("Finders of fact are entitled—indeed, expected—to make determinations as to the reliability of the evidence presented to them and, if appropriate, as to the credibility of the persons presenting that evidence."); Althen, 418 F.3d at 1280 (noting that "close calls" are resolved in Petitioner's favor).

"Testimony that merely expresses the possibility—not the probability—is insufficient, by itself, to substantiate a claim that such an injury occurred. See Waterman v. Sec'y of Health & Hum. Servs., 123 Fed. Cl. 564, 573-74 (2015) (denying a petitioner's motion for review and noting that a possible causal link was not sufficient to meet the preponderance standard). The Federal Circuit has made clear that the mere possibility of a link between a vaccination and a petitioner's injury is not sufficient to satisfy the preponderance standard. Moberly, 592 F.3d at 1322 (emphasizing that "proof of a 'plausible' or 'possible' causal link between the vaccine and the injury" does not equate to proof of causation by a preponderance of the evidence); Boatmon v. Sec'y of Health & Hum. Servs., 941 F.3d 1351, 1359-60 (Fed. Cir. 2019). While certainty is by no means required, a possible mechanism does not rise to the level of preponderance. Moberly, 592 F.3d at 1322; see also de Bazan, 539 F.3d at 1351.

IV. ANALYSIS

A. Causation

1. Althen Prong One

Under Althen prong one, Petitioner must set forth a medical theory explaining how the received vaccine could have caused the sustained injury. Andreu, 569 F.3d at 1375; Pafford, 451 F.3d at 1355-56. Petitioner's theory of causation need not be medically or scientifically certain, but it must be informed by a "sound and reliable" medical or scientific explanation. Boatmon, 941 F.3d 1351, 1359 (Fed. Cir. 2019); see also Knudsen, 35 F.3d at 548; Veryzer v. Sec'y of Health & Hum. Servs., 98 Fed. Cl. 214, 257 (2011) (noting that special masters are bound by both § 13(b)(1) and Vaccine Rule 8(b)(1) to consider only evidence that is both "relevant" and "reliable"). If Petitioner relies upon a medical opinion to support his theory, the basis for the opinion and the reliability of that basis must be considered in the determination of how much weight to afford the offered opinion. See Broekelschen v. Sec'y of Health & Hum. Servs., 618 F.3d 1339, 1347 (Fed. Cir. 2010) ("The special master's decision often times is based on the credibility of the experts and the relative persuasiveness of their competing theories."); Perreira v. Sec'y of Health & Hum. Servs., 33 F.3d 1375, 1377 n.6 (Fed. Cir. 1994) (stating that an "expert opinion is no better than the soundness of the reasons supporting it" (citing Fehrs v. United States, 620 F.2d 255, 265 (Ct. Cl. 1980))).

For the following reasons, the undersigned finds Petitioner has provided by preponderant evidence a sound and reliable theory by which the Prevnar 13 vaccine can cause GBS, and therefore, Petitioner has satisfied the first Althen prong.

Molecular mimicry has long been invoked as the causal mechanism for many different autoimmune diseases, including GBS. Many of the articles filed in this case support the mechanism as a leading hypothesis for the etiology of GBS. The theory has been extended from infectious agents to vaccine-associated autoimmune illnesses, including GBS.

Molecular mimicry has been accepted as a sound and reliable theory in many demyelinating conditions, including GBS, in the Vaccine Program, forming the basis for petitioners to be entitled to compensation. See, e.g., Conte v. Sec'y of Health & Hum. Servs., No. 17-403V, 2020 WL 5743696, at *57 (Fed. Cl. Spec. Mstr. July 27, 2020) (noting the theory of molecular mimicry in a GBS case is "well-established and well-settled in the Vaccine Program"); Barone v. Sec'y of Health & Hum. Servs., No. 11-707V, 2014 WL 6834557, at *8-9 (Fed. Cl. Spec. Mstr. Nov. 12, 2014) (noting molecular mimicry "has been accepted in other Program cases as a reliable medical explanation for how various autoimmune conditions could develop after the receipt of different kinds of vaccinations"); Koller, 2021 WL 5027947, at *18; Pierson v. Sec'y of Health & Hum. Servs., No. 17-1136V, 2022 WL 322836, at *31 (Fed. Cl. Spec. Mstr. Jan. 19, 2022); Maloney v. Sec'y of Health & Hum. Servs., No. 19-1713V, 2022 WL 1074087 (Fed. Cl. Spec. Mstr. Mar. 17, 2022); Gross v. Sec'y of Health & Hum. Servs., No. 17-

1075V, 2022 WL 9669651 (Fed. Cl. Spec. Mstr. Sept. 22, 2022); Sprenger v. Sec'y of Health & Hum. Servs., No. 18-279V, 2023 WL 8543435, *18-20 (Fed. Cl. Spec. Mstr. Nov. 14, 2023).[45]

Dr. Callaghan suggests that the traditional criteria (cited by Dr. Gershwin) must be met to establish whether a vaccine can cause GBS via molecular mimicry. The first criterion is similarity between a host epitope and an epitope of the vaccine. Dr. Gershwin provided an example of homology between a host epitope (phospholipids in myelin) and an epitope in two serotypes in the vaccine (18C and 23F). The second criterion is "detection of antibodies." Dr. Gershwin provided evidence of antibodies in patients with GBS by citing to Gilburd et al.

Admittedly, there is a lack of epidemiological evidence other than the Haber et al. article and two case reports. And there are no animal models to support Dr. Gershwin's theory. Therefore, the traditional criteria used to prove molecular mimicry are not fulfilled, as readily admitted by Dr. Gershwin. However, given the state of current scientific knowledge, it would not be possible for a petitioner to satisfy these criteria. Further, fulfilment of these criteria would require scientific certainty, which is a bar too high. See Knudsen, 35 F.3d at 549 (explaining that "to require identification and proof of specific biological mechanisms would be inconsistent with the purpose and nature of the vaccine compensation program").

Dr. He's reliance on epidemiology studies to opine that the Prevnar 13 vaccine does not cause GBS falls short of the mark. A careful review of the studies he cited establishes that the Prevnar 13 vaccine was not studied, or if it was, there was insufficient information on which to reach any conclusions. Haber et al. appears to be the only study cited specific to Prevnar 13, and while the authors stated that the incidence of GBS was not disproportionate, the data and methodology used to reach that conclusion was not provided. Thus, there is a lack of epidemiological information one way or the other on the question of a causal association between Prevnar 13 and GBS. Further, Petitioner does not need to make a specific type of evidentiary showing (i.e., epidemiologic studies) to satisfy his burden. See Capizzano, 440 F.3d at 1325-26.

Moreover, Dr. He does not refute the scientific data or foundational evidence used by Dr. Gershwin to support his theory. Dr. Gershwin identified components of the vaccine that could initiate development of antibodies that could cross-react with epitopes on peripheral nerve myelin or axonal glycoproteins. He also identified components of the Prevnar 13 vaccine that could trigger a human antibody response.

Regarding Petitioner's theory based on phosphoglycerol in serotypes 18C and 23F in the vaccine, Dr. Gershwin produced a paper to show that in multiple sclerosis, myelin phospholipids are targeted by an immune response. He also showed that myelin is comprised of phospholipids, and that phospholipids can serve as autoantigens in autoimmune disorders. He further showed that patients with GBS have autoantibodies to phospholipids. In summary, there is sound support from reputable medical studies for the foundational aspects of Dr. Gershwin's phosphoglycerol theory.

[45] The undersigned acknowledges that the first two cases in this string cite involve a different vaccine, although the same illness.

Additionally, the causal theory proffered by Petitioner has previously been accepted as sound and reliable in other cases, decided by various special masters, including the undersigned. See, e.g., Sprenger, 2023 WL 8543435; Gross, 2022 WL 9669651; Maloney, 2022 WL 1074087; Pierson, 2022 WL 322836; Koller, 2021 WL 5027947. While prior decisions are not binding on the undersigned, they can be considered by the undersigned in forming her opinions. See Hanlon v. Sec'y of Health & Hum. Servs., 40 Fed. Cl. 625, 630 (1998), aff'd, 191 F.3d 1344 (Fed. Cir. 1999); Boatmon, 941 F.3d at 1358. The undersigned agrees with the reasoning offered by her colleagues in these other cases, and for many of the same reasons finds Petitioner's theory here sound and reliable and proven by preponderant evidence.

The undersigned recognizes that there is not uniformity between the special masters in decisions addressing the Prevnar 13 vaccine and GBS. See, e.g., Deshler v. Sec'y of Health & Hum. Servs., No. 16-1070V, 2020 WL 4593162, at *19 (Fed. Cl. Spec. Mstr. July 1, 2020); Trollinger v. Sec'y of Health & Hum. Servs., No. 16-473V, 2023 WL 2521912, at *26 (Fed. Cl. Spec. Mstr. Feb. 17, 2023), mot. for review denied, 167 Fed. Cl. 127; Bielak v. Sec'y of Health & Hum. Servs., No. 18-761V, 2023 WL 35509, at *31-32 (Fed. Cl. Spec. Mstr. Jan. 3, 2023); Gamboa-Avila v. Sec'y of Health & Hum. Servs., No. 18-925V, 2023 WL 6536207, at *25 (Fed. Cl. Spec. Mstr. Sept. 11, 2023); McConnell v. Sec'y of Health & Hum. Servs., No. 18-1051V, 2022 WL 4008238, at *9 (Fed. Cl. Spec. Mstr. Aug. 19, 2022). The undersigned acknowledges these cases but also notes that the decisions of other special masters or Court of Federal Claims' judges are not binding on special masters. Boatmon, 941 F.3d at 1358; Hanlon, 40 Fed. Cl. at 630.

For these reasons, the undersigned finds that Petitioner has proven by preponderant evidence a sound and reliable causal theory establishing that the Prevnar 13 vaccine can cause GBS, satisfying Althen prong one.

2. Althen Prong Two

Under Althen prong two, Petitioner must prove by a preponderance of the evidence that there is a "logical sequence of cause and effect showing that the vaccination was the reason for the injury." Capizzano, 440 F.3d at 1324 (quoting Althen, 418 F.3d at 1278). "Petitioner must show that the vaccine was the 'but for' cause of the harm . . . or in other words, that the vaccine was the 'reason for the injury.'" Pafford, 451 F.3d at 1356 (internal citations omitted).

In evaluating whether this prong is satisfied, the opinions and views of the vaccinee's treating physicians are entitled to some weight. Andreu, 569 F.3d at 1367; Capizzano, 440 F.3d at 1326 ("[M]edical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a 'logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.'" (quoting Althen, 418 F.3d at 1280)). Medical records are generally viewed as trustworthy evidence, since they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528. While the medical records and opinions of treating physicians must be considered, they are not binding on the special master. § 13(b)(1)(B) (specifically stating that the "diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court").

A petitioner need not make a specific type of evidentiary showing, i.e., "epidemiologic studies, rechallenge, the presence of pathological markers or genetic predisposition, or general acceptance in the scientific or medical communities to establish a logical sequence of cause and effect." Capizzano, 440 F.3d at 1325. Instead, Petitioner may satisfy his burden by presenting circumstantial evidence and reliable medical opinions. Id. at 1325-26.

The undersigned finds there is preponderant evidence of a logical sequence of cause and effect establishing that the Prevnar 13 vaccination administered to Petitioner was the cause of his GBS. First, Petitioner was appropriately diagnosed with GBS, and he has offered preponderant evidence of a sound and reliable mechanism of vaccine causation.

Second, Petitioner's clinical course was consistent with post-vaccination GBS. Dr. Frey opined that Petitioner's clinical course was consistent with GBS, and that his diagnosis was confirmed by neurological examination and EMG. Improvement with IVIG also confirms Petitioner's diagnosis and the fact that he had an autoimmune illness. These opinions were not rebutted by Respondent's experts.

Petitioner received his Prevnar 13 vaccination on April 4, 2017. On April 17, 2017, Petitioner presented to his PCP reporting four days of progressive, ascending numbness, tingling, and weakness. Petitioner reported that the symptoms began in his right heel and progressed to include both of his legs, arms, tongue, and cheeks. On physical examination, he had paresthesias bilaterally in his upper extremities and lower extremities, weakness in both calves, and an abnormal gait. Petitioner's condition worsened, and on April 25, he was taken to the Mayo Clinic Hospital ER. Upon arrival, Petitioner reported a 10-day history of weakness, upper extremity paresthesias, and voice changes. He reported falling and developing slurred speech and difficulty walking. Physical examination revealed that his deep tendon reflexes were absent in his patellar and Achilles tendons. The impression was weakness and paresthesias of unclear etiology. EMG showed sensorimotor polyradiculoneuropathy with primarily demyelinating features. Petitioner was diagnosed with GBS. He was treated with five rounds of IVIG and started on Gabapentin for pain. Petitioner received rehabilitation and has not had any reoccurrence of his GBS, although he continues to experience sequelae from his illness.

The Qualification and Aids to Interpretation ("QAI") of the Vaccine Injury Table relevant to post-vaccination GBS for the seasonal influenza vaccination requires five criteria for inclusion as a Table Injury. Although the Table criteria are specific to the seasonal influenza vaccination, they can be used as surrogate criteria here to describe post-vaccination GBS in the context of Prevnar 13. The QAI criteria include:

(A) Bilateral flaccid limb weakness and decreased or absent deep tendon reflexes in weak limbs;
(B) A monophasic illness pattern;
(C) An interval between onset and nadir of weakness between 12 hours and 28 days;

(D) Subsequent clinical plateau (the clinical plateau leads to either stabilization at the nadir of symptoms, or subsequent improvement without significant relapse . . .); and,
(E) The absence of an identified more likely alternative diagnosis.

42 C.F.R. § 100.3(c)(15)(iii).

Petitioner's clinical course was consistent with all the above criteria. He developed bilateral limb weakness in his legs, followed by absent deep tendon reflexes. He had a monophasic illness pattern, meaning that his illness occurred one time, and he did not experience any significant relapse or reoccurrence. It is difficult to determine the time interval between onset and nadir of Petitioner's weakness, but his condition worsened to the point that he was taken by ambulance to the Mayo Clinic Hospital on April 25, approximately 12 days after onset, which is within the 28 days specified by the criteria. Petitioner also improved over time, and he experienced subsequent improvement without significant relapse, although he continues to have side effects from his GBS.

Next, there must not be a "more likely alternative diagnosis." 42 C.F.R. § 100.3(c)(15)(iii)(E). Petitioner's medical records, physician notes, and diagnostic workup did not reveal an infectious or alternate cause of Petitioner's GBS. And Respondent's experts did not identify or propose any alternative cause of Petitioner's GBS. The only antecedent event present to trigger an immune response was the Prevnar 13 vaccination administered to Petitioner on April 4, 2017.

Further, the Vaccine Table finds diagnostic evidence supportive. 42 C.F.R. § 100.3(c)(15)(iv). Here, Petitioner underwent an EMG that was consistent with GBS.

In addition to the criteria discussed above, the Table identifies a long list of exclusionary criteria, including but not limited to chronic immune demyelinating polyradiculopathy, carcinomatous meningitis, myelitis, spinal cord infarct, West Nile virus, multiple sclerosis, and others. 42 C.F.R. § 100.3(c)(15)(vi). Petitioner did not have any of the conditions identified in the list. He was not diagnosed with any other disorder which could have caused the symptoms that were attributed to his GBS.

Third, Petitioner's treating neurologist, Dr. Grill offered a supportive causal opinion. Dr. Grill provided medical care to Petitioner after his hospitalization and inpatient rehabilitation. Petitioner saw Dr. Grill on July 26, 2017, about two months after his discharge from rehabilitation. Dr. Grill documented, "With respect to the causative agent regarding his [GBS], there was no antecedent illness noted though he did have a vaccination prior; thus, this certainly could have been the medium to prompt this immune reaction." Pet. Ex. 8e at 364. The undersigned finds Dr. Grill's note expressing her opinion as to causation to be evidence of a causal association between Petitioner's Prevnar 13 vaccination and the development of his GBS. Further, the opinions of treating physicians are generally more reliable because they are created contemporaneously with the treatment of the vaccinee. Cucuras, 993 F.2d at 1528.

In summary, Petitioner's medical records, physician notes, and diagnostic workup reveals a clinical course consistent with GBS as described in the Vaccine Injury Table. Further, there is no evidence of an infectious or alternate cause of Petitioner's GBS. The only reference to an antecedent event was related to Petitioner's vaccination on April 4, 2017.

In conclusion, the undersigned finds that Petitioner has proven by preponderant evidence a logical sequence of cause and effect establishing that the Prevnar 13 vaccination caused his GBS and has satisfied the second Althen prong.

3. Althen Prong Three

Althen Prong three requires Petitioner to establish a "proximate temporal relationship" between the vaccination and the injury alleged. Althen, 418 F.3d at 1281. That phrase has been defined as a "medically acceptable temporal relationship." Id. Petitioner must offer "preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." de Bazan, 539 F.3d at 1352. The explanation for what is a medically acceptable time frame must also coincide with the theory of how the relevant vaccine can cause the injury alleged (under Althen prong one). Id.; Koehn v. Sec'y of Health & Hum. Servs., 773 F.3d 1579, 1243 (Fed. Cir. 2014); Shapiro, 101 Fed. Cl. at 542 (2011); see also Pafford, 451 F.3d at 1358.

Respondent's experts do not disagree that there is a temporal association between Petitioner's vaccination and onset of GBS. Petitioner received his Prevnar 13 vaccination on April 4, 2017, and onset was approximately nine days later, on April 13, 2017. The time frame of nine days from vaccination to the initial manifestation of symptoms is appropriate given the theory of molecular mimicry, and as demonstrated in Haber et al. In Haber et al., 11 cases of GBS following a Prevnar 13 vaccine were reported, and the median onset interval was nine days. This temporal association is also consistent with the onset period of three to 42 days as set forth in the Vaccine Injury Table for GBS following influenza vaccination. 42 C.F.R. § 100.3(a)(XIV)(D).

Further, this time frame has been acknowledged as appropriate in other Vaccine Program cases in which molecular mimicry has been proffered as the causal mechanism. See, e.g., Maloney, 2022 WL 1074087, at *36 (finding a GBS onset of seven days after Prevnar 13 vaccination to be appropriate); Koller, 2021 WL 5027947, at *57 (finding a GBS onset of 12 days after Prevnar 13 vaccination to be "within the medically accepted timeframe consistent with [P]etitioner's theory of molecular mimicry [and] that has been accepted in other Vaccine Program cases."); Barone, 2014 WL 6834557, at *13 ("[S]pecial masters have never gone beyond a two-month (meaning eight week) interval in holding that a vaccination caused a demyelinating illness."); Gross, 2022 WL 9669651, at *38-39 (finding a GBS onset of 13 days after Prevnar 13 vaccination to be appropriate); Sprenger, 2023 WL 8543435, at *22 (finding a GBS onset of two weeks post-Prevnar 13 vaccination to be appropriate given the theory of molecular mimicry).

Therefore, undersigned finds that Petitioner has met his burden of proof as to Althen prong three.

V. CONCLUSION

Based on the record, and for the reasons discussed above, the undersigned finds there is preponderant evidence to satisfy all three Althen prongs and to establish that Petitioner's Prevnar 13 vaccination caused his GBS. Thus, the undersigned finds that Petitioner is entitled to compensation. A separate damages order will issue.

IT IS SO ORDERED.

s/Nora Beth Dorsey
Nora Beth Dorsey
Special Master

Summary

Congress passed the National Childhood Vaccine Injury Act (P.L. 99-660) in 1986. The legislation was intended to bolster vaccine research and development through federal coordination of vaccine efforts in government by providing relief to vaccine manufacturers who reported at the time that financial burdens from awards in the tort system threatened their financial viability. The legislation was also intended to address concerns about the safety of vaccines through a multipronged approach involving instituting a compensation program financed by an excise tax on covered vaccines, setting up a passive surveillance system for vaccine adverse events, and providing information to consumers.

Sections 312 and 313 of the legislation required the secretary of the U.S. Department of Health and Human Services to consult with the Institute of Medicine (IOM) to conduct a review of the scientific literature related to a set of serious adverse events[1] following immunizations recommended for use in children. Two reports were issued (IOM, 1991, 1994). These reports contain a framework for causality assessment of adverse events following vaccination. The reports embraced all vaccines covered by the National Vaccine Injury Compensation Program (VICP) up to that point: diphtheria- and tetanus-toxoids and whole cell pertussis (DTwP) vaccine[2] and other tetanus toxoid–containing vaccines; measles, mumps, and rubella

[1] Adverse *events* are distinguished from adverse *effects* in that an event is something that occurs but may not be causally associated, whereas an adverse *effect* implies causation. All adverse effects are adverse events, but not all adverse events are adverse effects.

[2] Acellular pertussis vaccine (aP) has replaced whole cell pertussis vaccine in the United States.

(MMR) vaccines; *Haemophilus influenzae* type B vaccine; hepatitis B vaccine; and both inactivated and oral polio vaccines.[3] The reports informed the secretary's review of the Vaccine Injury Table. The reports have also been referenced extensively as a source of definitive scientific understanding of the evidence by Special Masters in decisions regarding injuries not listed on the Vaccine Injury Table.

The IOM was subsequently asked to review specific vaccine safety concerns in a series of reports requested by the Centers for Disease Control and Prevention (CDC). These reports (IOM, 2001a,b, 2002a,b, 2003a,b, 2004a,b) included causality assessments similar to the previous IOM reports, but included other conclusions and recommendations regarding research, communications, and policy review.

CHARGE TO THE COMMITTEE

In 2009 the IOM entered into a contract with the Health Resources and Services Administration (HRSA)[4] to convene a committee of experts to review the epidemiologic, clinical, and biological evidence regarding adverse health events associated with specific vaccines covered by the VICP. The committee was composed of individuals with expertise in pediatrics, internal medicine, neurology, immunology, immunotoxicology, neurobiology, rheumatology, epidemiology, biostatistics, and law.

The vaccines to be reviewed included varicella zoster vaccine; influenza vaccines;[5] hepatitis B vaccine; human papillomavirus vaccine (HPV); tetanus toxoid–containing vaccines other than those containing the whole cell pertussis component; measles, mumps, and rubella vaccines; hepatitis A vaccine; and meningococcal vaccines. It is expected that the report will provide the scientific basis for review and adjudication of claims of vaccine injury by the VICP.

HRSA presented a list of specific adverse events for the committee to review (see Table S-1). The selection criteria was described at the first committee meeting (Johann-Liang, 2009) as including the vast majority of adverse events in the claims for compensation. The committee added adverse events to the list if it identified epidemiologic studies or case reports for an adverse event not originally assigned by HRSA. These additions were all-cause mortality and seizures following influenza vaccine; optic neuritis

[3] Vaccines are included in the VICP if they are recommended by the CDC for routine administration in children and are subject to an excise tax. Adults who experience an adverse reaction to one of these "childhood" vaccines are also covered by the program.

[4] The CDC and the National Vaccine Program Office also provided funds for the project via the contract with HRSA.

[5] The 2009 H1N1 influenza vaccine is covered by the Countermeasures Injury Compensation Program, and evidence about its safety is not covered in this report.